detectable AWM was significantly greater in the Q compared with the non-Q group ($p < 0.001$). When those non-Q patients without AWM are excluded, there was no significant difference in the extent of AWM between the Q and non-Q groups. Among those patients with AWM at entry who underwent repeat echocardiography at 6–12 weeks (final), both the Q and non-Q groups showed a significant reduction in extent of AWM. However, the mean % change (Δ) in AWM was significantly greater in the non-Q than in the Q group, and there was a trend toward lower final extent of AWM in the non-Q group. The mean ESAi was not significantly different between the two groups at entry or at follow up.

	Q (n = 41)	Non-Q (n = 7)	p
AWM entry	29 ± 19*	24 ± 11**	0.53
AWM final	22 ± 21*	8 ± 11**	0.10
Mean % Δ AWM	−22 ± 55	−75 ± 28	<0.02

*$p < 0.02$, **$p < 0.002$

These findings were independent of the performance of angioplasty. In conclusion, failure to develop Q waves following thrombolysis predicts a lower likelihood of developing significant regional LV dysfunction and, if dysfunction is present, predicts a greater degree of recovery.

4:45

806-4 Association of Ventricular Arrhythmias with Left Ventricular Remodeling After Myocardial Infarction: Is it a Missing Link?

Aleksandar D. Popović, Kočo Pavlovski, Aleksandar N. Nešković, Jelena Marinković, Rade Babić, James D. Thomas *Clinical-Hospital Center Zemun, Belgrade University Medical School, Belgrade, YU; Cleveland Clinic Foundation, Cleveland, OH*

It is well known that both complex ventricular arrhythmias (CVA) and LV dysfunction after MI are predictors of poor prognosis. Several studies have assessed the relationship of CVA and LVEF with controversial results; however, the relationship of CVA and LV remodeling after MI has not been clarified yet. We have prospectively evaluated 97 consecutive pts with acute MI by serial echocardiographic examinations (day 1, 2, 3 and 7 and after 3 weeks) in order to determine end-diastolic volume index (EDVi), end-systolic volume index (ESVi) and EF. Holter monitoring was performed after 3 weeks. Coronary angiography was performed in 88 patients before hospital discharge.

Results: CVA (defined as Lown classes 3–5) were found in 16/97 pts. In the logistic regression model, variables predictive of CVA were ESVi on admission ($b = 0.048$, $p = 0.032$) and EDVi after 3 weeks ($b = 0.034$, $p = 0.012$). CVA were related to the increase of EDVi and ESVi throughout the study ($F = 5.62$, $p = 0.046$ and $F = 6.42$, $p = 0.017$, respectively by MANOVA). The incidence of CVA was not related to EF, thrombolysis, infarct-related artery patency, angiographic extent of coronary artery disease and infarct location.

Conclusions: These data indicate that CVA are related to progressive LV dilation, rather than to depressed EF. It appears that CVA may be the missing link that explains association of LV remodeling with higher mortality.

807 Cardiac Function and Failure in the Elderly

Wednesday, March 22, 1995, 4:00 p.m.–5:00 p.m.
Ernest N. Morial Convention Center, Room 16

4:00

807-1 Congestive Heart Failure with Preserved Systolic Function in a Large Community-Dwelling Elderly Cohort: The Cardiovascular Health Study

Julius M. Gardin, Alice Arnold, Dalane Kitzman, Vivienne E. Smith, Joao A.C. Lima, H. Sidney Klopfenstein, Diane E. Bild, CHS Research Group. *University of California, Irvine, CA*

LV diastolic dysfunction is known to be an important cause of congestive heart failure (CHF) in the elderly. However, the prevalence of LV diastolic dysfunction as a mechanism of CHF in a large, elderly cohort is unknown. The Cardiovascular Health Study is an NHLBI sponsored multi-center study of community-dwelling individuals 65 years and older designed to evaluate cardiovascular risk, mortality and morbidity. In Year 2 (1989–90), 4,629 of 5,201 individuals successfully underwent two-dimensional echocardiography (2-D echo) evaluation of the left ventricle (LV). The table outlines the prevalence of definite CHF by history and LV systolic function as assessed by 2-D echo. *Normal* LV systolic function was defined as normal LV ejection fraction (EF) and wall motion by qualitative/semiquantitative assessment, *abnormal* was defined as presence of either abnormal EF or wall motion (akinesis/dyskinesis), and *borderline* was intermediate.

	Overall	Men	Women
Total Studied by Echo	4,629	1,971	2,658
Definite CHF by History	79 (1.7%)	43 (2.2%)	36 (1.4%)
Abnormal LV Systolic Function	31 (39%)	20 (46%)	11 (31%)
Borderline LV Systolic Function	11 (14%)	5 (12%)	6 (17%)
Normal LV Systolic Function	37 (47%)	18 (42%)	19 (53%)

Conclusions: In this large elderly cohort: (1) the prevalence of definite congestive heart failure by history was 2%; (2) nearly one-half of participants with definite CHF had normal LV systolic function; (3) the distribution of systolic dysfunction among participants with definite CHF did not differ significantly by gender. These findings suggest a high prevalence of LV diastolic dysfunction as the mechanism of CHF among a large cohort of elderly, community-dwelling individuals.

4:15

807-2 Effect of Age on Left Ventricular Diastolic Filling Patterns During Orthostatic Stress

Christian J. Swinne, Edward P. Shapiro, Frances C. O'Connor, Patricia Reyerson, Jerome L. Fleg *Gerontology Research Center, NIA, NIH; Johns Hopkins Bayview Medical Center, Baltimore, MD*

Although numerous studies have demonstrated reduced early diastolic left ventricular (LV) peak filling velocity (E) and accentuated late filling velocity (A) with advancing age in the supine position, the effect of orthostatic stress on age-associated diastolic filling patterns is unknown. Accordingly, 30 healthy normotensive volunteers ages 19 to 90 years from the Baltimore Longitudinal Study of Aging underwent sequential Doppler echocardiography after 3 minutes each in the supine, seated, and standing positions. In the overall sample, standing was accompanied by an increase in heart rate (HR) of 8.8 ± 1.7 beats/min, and decreases in E (20.6 ± 2.7 cm/s), A (5.0 ± 2.7 cm/s)and LV diastolic dimension (LVDD) (7.8 ± 0.8 mm), $\bar{x} \pm$ SEM, each $p < 0.001$ versus supine values. Systolic blood pressure (SBP) and atrial filling fraction (AFF) were unaffected by posture. Correlation coefficients versus age for relevant Doppler and hemodynamic variables are shown.

	E	A	E/A	AFF	HR	SBP	LVDD
Supine	−0.58†	0.80†	−0.78†	0.84†	0.01	0.56†	−0.14
Sit	−0.36	0.77†	−0.83†	0.78†	−0.04	0.37*	−0.07
Stand	−0.24	0.60†	−0.69†	0.71†	−0.32	0.47†	0.09
ΔSupine→Stand	0.53†	−0.46†	0.56†	−0.17	−0.61†	0.22	0.40*

*$p < 0.05$ †$p < 0.01$

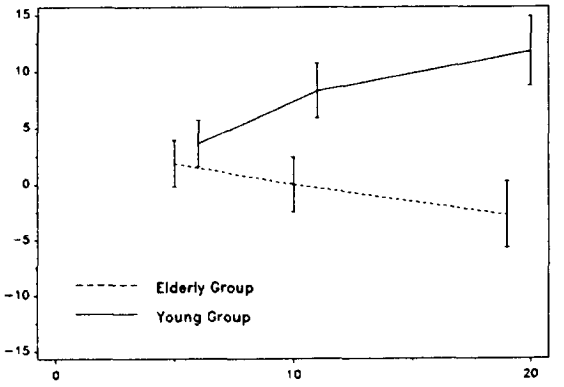
Thus, orthostatic stress abolishes the reduction of E and attenuates the exaggerated A observed with advancing age in the supine position, probably because of the blunted standing-induced HR increase in older subjects. However, the supine age-associated increase in AFF is unaffected by orthostasis.

4:30

807-3 The Effect of Age on the Hemodynamic Response to Dobutamine

L. Ashley Stroud, Gregory B. Russell, Dalane W. Kitzman. *Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, North Carolina*

Although aging is associated with decreased beta-adrenergic responsiveness and many patients undergoing dobutamine stress echocardiography (DSE) are elderly, the effect of age on the hemodynamic response to dobutamine (DOB) has not been reported. Furthermore, hypotension is frequently observed during DSE and the mechanism is controversial. Therefore, we examined the heart rate (HR) and systolic blood pressure (SBP) responses in



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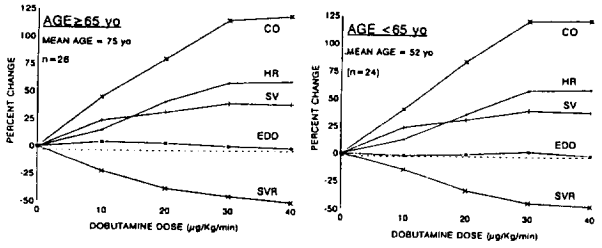
2 gender-matched groups of subjects widely separated in age by 2 decades: an older group (age 73 ± 4, n = 42) and a younger group (age 53 ± 7, n = 41), during graded 3 minute infusions of DOB at 5, 10, and 20 μg/kg/min. We excluded subjects who were on cardioactive medications or who had abnormal LV function or conduction defects. *Results:* HR responses of the two groups were nearly identical at all doses. In contrast, SBP responses differed markedly (see figure). With increasing DOB dose, the young had an increase in SBP while the old had a progressive decrease in SBP ($p < 0.01$ by ANOVA), even after adjusting for higher resting SBP in the old. The SBP response was not affected by wall motion or LV function. *Conclusion:* Aging markedly alters the blood pressure but not the heart rate response to DOB. These data may help explain the frequency of hypotension during DSE.

4:45

807-4 Normal Hemodynamic Responses to Dobutamine Infusion in the Elderly

Craig Lampert, Herman D. Movsowitz, Larry E. Jacobs, Alfred Ioli, Morris N. Kotler
Albert Einstein Medical Center, Philadelphia, PA

Elderly patients undergoing conventional exercise stress testing (CST) rely predominantly on an increased preload (Starling mechanism) to augment cardiac output (CO). In contrast, younger patients rely predominantly on an increased heart rate (HR) and decreased systemic vascular resistance (SVR) to augment CO. To determine normal physiologic hemodynamic responses to incremental dobutamine infusion in young (<65 yo) as compared to elderly (≥65 yo) patients, we analyzed the results of 50 consecutive patients with normal regional wall motion and normal ejection fraction at rest. All patients underwent 2-D echocardiography and continuous wave Doppler evaluation of the left ventricular outflow tract (LVOT) during each stage of the dobutamine infusion protocol. No patients developed regional wall motion abnormalities during dobutamine infusion. HR, velocity time integral (VTI) across the LVOT and end diastolic dimensions (EDD) were recorded. Stroke volume (SV = VTI × cross sectional area of LVOT), CO (HR × SV) and SVR (80 × mean arterial pressure ÷ CO) were calculated. The mean % change from baseline of HR, SV, EDD, SVR and CO are shown:



Conclusion: 1) There is no statistical difference in the hemodynamic response to dobutamine in young versus elderly patients. 2) Elderly patients undergoing dobutamine stress testing augment their CO by increasing HR and SV, decreasing SVR and with no change in EDD. 3) This is in contrast to that observed during CST. 4) Adequate heart rate response in the elderly might be more important during dobutamine infusion than during CST.

808 Electrophysiology — Basic Ventricular Arrhythmias

Wednesday, March 22, 1995, 4:00 p.m.–5:00 p.m.
Ernest N. Morial Convention Center, Room 14

4:00

808-1 Focal Mechanisms Underlying Sustained Ventricular Tachycardia in Ischemic Cardiomyopathy

Carolyn M. Johnson, Steven Pogwizd *Washington University, St. Louis, MO*

To define the mechanism of ventricular tachycardia (VT) induced by programmed electrical stimulation in the setting of congestive heart failure, 3-dimensional cardiac mapping was performed in six dogs with ischemic cardiomyopathy induced by multiple intracoronary embolizations with microspheres. Ejection fraction (EF) progressively decreased from 54 ± 2 to 30 ± 5% ($p < 0.005$) after an average of 6 weekly embolizations. Four months later (EF = 28 ± 5%), each dog underwent a thoracotomy with insertion of plunge-needle electrodes into the heart under pentobarbital anesthesia. Continuous recording from 232 intramural sites throughout the left and right ventricles and the interventricular septum was performed during spontaneous rhythm and during programmed stimulation in both the absence and presence of isoproterenol (Iso) (0.1 mg/kg/min). Three dogs (1 at baseline, 2 during Iso) developed sustained VT (SuVT) during programmed stimulation with up to 3 extrastimuli. After the last extrastimulus, the first beat of SuVT (T_1) initiated in the subendocardium by a focal mechanism, based on the absence of electrical activity from the termination of the last extrastimulus to the initiation of T_1 despite the presence of multiple intermediate electrode sites. Maintenance of SuVT was also due to a focal mechanism arising in the subendocardium, with a total activation time (TAT) of subsequent SuVT beats (99 ± 14 msec, n = 6) that was comparable to that of initiating beats (85 ± 8 msec, n = 6, p = 0.08). There was no evidence of macroreentry. Episodes of SuVT (n = 3) exhibited TATs of the last extrastimulus before SuVT (106 ± 11 msec) and coupling intervals of T_1 (196 ± 70 msec) that were comparable to those observed in nonsustained VT (n = 3) (138 ± 26 msec, p = 0.19; 166 ± 26 msec, p = 0.59). Conduction delay during sinus rhythm in dogs with SuVT (TAT 47 ± 5 msec, n = 10) was comparable to that in dogs without SuVT (TAT 47 ± 4 msec, n = 10, p = 0.96) and was unchanged by Iso (TAT 50 ± 4 msec, n = 7, p = 0.40). In the 2 dogs with inducible SuVT on Iso only, TATs of the last extrastimulus (124 ± 22 msec) and of T_1 (110 ± 36 msec) at baseline were unchanged by infusion of Iso (108 ± 13 msec, p = 0.59; 79 ± 1 msec, p = 0.49). Thus, inducible sustained VT in a model of ischemic cardiomyopathy is due to a focal mechanism, as opposed to macroreentry, and this focal mechanism is enhanced by β-adrenergic stimulation.

4:15

808-2 Sudden Heart Rate Speeding and Slowing Facilitates the Inducibility of Ventricular Tachycardia in Dogs

Tadashi Satoh, Harold P. Pride, Douglas P. Zipes. *Krannert Institute of Cardiology, Indiana University School of Medicine, Indianapolis, IN*

Clinically, torsades de pointes (TdP) often occurs when the heart rate suddenly slows (eg. atrial fibrillation to sinus rhythm). We investigated whether the dose of cesium chloride (CsCl) required to induce early afterdepolarizations (EADs), the putative mechanism of TdP, and ventricular tachycardia (VT) was different in dogs with a paced left ventricular cycle length (PCL) of 1,000 msec for a 1 week versus dogs with a PCL of 1,000 msec for 1 week and 500 msec for 1 hour prior to a PCL of 1,000 msec. All dogs had atrioventricular (AV) block induced by radiofrequency ablation and were studied closed chest. While recording surface ECG leads I,II,III and LV endocardial monophasic action potential (MAP), CsCl was injected incrementally (0.25, 0.5, 0.625, 0.75, 1.0 mM/kg) until sustained VT was induced. In group 1 (n = 6), CsCl was injected during PCL = 1,000 msec. In group 2 (n = 7), CsCl was injected during PCL = 1,000 msec after PCL = 500 msec for 1 hour. In group 1, VT was induced at 0.75 mM/kg in 3 dogs and at 1.0 mM/kg in 3 dogs. VT was induced at 0.75 mM/kg in all 7 dogs in group 2 ($p < 0.05$). The area of EAD as a percentage of LV MAP area (%EAD) in group 2 dogs exceeded that of group 1 at 0.5, 0.625, 0.75 mM/kg.

CsCl (mM/kg)	0.25	0.5	0.625	0.75
Group 1 (%EAD)	108 ± 2.4	115 ± 3.9	119 ± 5.4	129 ± 5.1
Group 2 (%EAD)	108 ± 3.6	118 ± 2.1	127 ± 3.9	135 ± 5.4
p	ns	<0.05	<0.01	<0.05

We conclude that only 1 hour of rapid pacing after 1 week of bradycardia is sufficient to produce a change in myocardial responsiveness to the K^+ channel blocker, CsCl, and increases the susceptibility of VT induction, possibly via an increase in EAD amplitude.

4:30

808-3 Reentrant Wavefronts During Wiggers' Stage II Ventricular Fibrillation in Dogs

John J. Lee, Dustan Hough, Chun Hwang, Wei Fan, Michael C. Fishbein, Claudio Bonometti, Hrayr S. Karaguezian, Peng-Sheng Chen *Cedars-Sinai Medical Center, and UCLA School of Medicine, Los Angeles, CA*

The mechanisms of ventricular fibrillation (VF) are unknown. Reentrant wavefronts have been shown to underlie the onset (Wiggers' stage I) of electrically induced VF in intact canine ventricles. These reentrant wavefronts, however, have a limited lifespan (1–2 s) while VF persists. Using computerized mapping techniques, we studied the mechanism by which VF is maintained beyond the initial few seconds (Wiggers' stage II), both in normal and subendocardium-ablated canine ventricles. Eleven open-chest dogs were studied. In 6 of the dogs, the RV subendocardium was ablated with Lugol's solution. A plaque electrode array with 317–509 bipolar recording electrodes was sutured on the RV epicardium. VF was induced by a strong premature stimulus (S_2). Starting 2.5 s after the onset of VF, 2–5 s of data were analyzed. The activation patterns were visualized via dynamic display. Conventional isochronal maps were also constructed. Of the 15 runs of VF in dogs with intact ventricles, 3 episodes of reentrant wavefronts were detected. The mean lifespan was 4.5 ± 2.1 rotations. The mean cycle length was 102.5 ±